In the United States Court of Federal Claims
OFFICE OF SPECIAL MASTERS
No. 23-1051V

SONIA BORGELT,	*	Chief Special Master Corcoran
Petitioner,	*	Filed: January 5, 2026
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Isaiah Kalinowski, Bosson Legal Group, Fairfax, VA, for Petitioner.

Rachelle Bishop, U.S. Department of Justice, Washington, DC, Respondent.

ENTITLEMENT DECISION[1]

On July 10, 2023, Sonia Borgelt filed a petition seeking compensation under the National Vaccine Injury Compensation Program (the "Vaccine Program").[2] Petitioner alleges that her receipt of an influenza ("flu") vaccine on September 10, 2020, significantly aggravated a "neurologic condition," which Petitioner now acknowledges was most likely a form of chronic inflammatory demyelinating polyneuropathy ("CIDP"). *See* Ex. 21 at 1.

A one-day Entitlement Hearing was held in Washington, D.C., on May 19, 2025. Now, for the reasons set forth below, and based on a complete review of the record, I deny entitlement. Petitioner has not preponderantly demonstrated that the flu vaccine can cause aggravation of the form of CIDP she experienced, or that it likely did so to her.

[1] Under Vaccine Rule 18(b), each party has fourteen (14) days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the whole Decision will be available to the public in its present form. *Id.*

[2] The Vaccine Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3758, codified as amended at 42 U.S.C. §§ 300aa-10 through 34 (2012) ("Vaccine Act" or "the Act"). Individual section references hereafter will be to § 300aa of the Act (but will omit that statutory prefix).

I. Factual History

Immediate Pre-Vaccination History

In August 2020—the month prior to the relevant vaccination—Petitioner (who was then 42) experienced some health issues bearing on her claim. Specifically, on August 17, 2020 (a little more than three weeks before the vaccination at issue), Ms. Borgelt went to the Providence Willamette Falls Medical Center ("PWF") Emergency Room ("ER") reporting back pain and numbness and tingling in her hands and feet. Ex. 3 at 442–46. She noted at this time her belief that the inciting incident was a minor fall she had experienced a week before, and the accident had been followed by back pain, and then upper back discomfort and a tingling and cold sensation in her feet—none of which had been successfully treated by medications prescribed by her primary care provider ("PCP"). *Id*. at 446.

On exam, Petitioner's lower back (mainly on the left side) displayed paraspinal muscle tenderness and spasms, although she had no neurologic deficits. *Id.* at 449. X-rays of her cervical and thoracic spine were normal, and a laboratory work-up revealed benign results. *Id*. at 450. Petitioner was diagnosed with acute back pain and numbness and tingling, prescribed pain relief medications and gabapentin (commonly used for nerve pain), and discharged home. *Id*. at 450–51.

A week later (August 24, 2020), Ms. Borgelt went to a different ER—at the Oregon Health & Science University ("OHSU")—for evaluation of a "fluttering" sensation in her chest that began that afternoon, plus spinal pain with numbness in her hands and feet. Ex. 6 at 192. She noted that she had increased her gabapentin, but then stopped taking it two days prior because it provided no relief. *Id*. Petitioner's chest x-ray and lab results were deemed normal. *Id*. at 192–201. She also had a telehealth visit with her PCP, Cristina De Castro-Dela Cruz, M.D., two days later, on August 26, 2020. Ex. 2 at 64. She reported her ER visits and further noted that she had been experiencing vaginal and perineal numbness but had control of her urination and bowel movements. *Id*. at 66. Dr. Cruz felt that Petitioner's existing x-ray imaging did not explain her upper extremity symptoms, and therefore ordered MRI studies of Petitioner's lumbar spine and brain—but the brain MRI was normal, and her cervical spine MRI showed enhancing annular fissuring at C5-C6 and C6-C7 levels, which were interpreted to "represent axial cervical pain generators in the proper clinical setting." *Id.* at 65; Ex. 3 at 117.

On August 27, 2020, Petitioner returned again to the ER, albeit at a different hospital (the Providence St. Vincent Medical Center ("St. Vincent")), reporting numbness in all extremities for the prior two weeks, plus lower back and intermittent chest pain. Ex. 4 at 690. A cardiac workup yielded normal results, however, and emergency medicine specialist Jeffrey Paul Lahti, M.D., found that Petitioner's head and neck MRI, CT scan, and ultrasound provided no explanation for her acute symptoms other than the evidence of degenerative changes at the C5-C7 spinal cord

levels. *Id*. at 694–95; *see also* Ex. 3 at 117. Dr. Lahti considered the possibility that Guillain-Barré syndrome ("GBS") was diagnostically explanatory, but deemed it "unlikely given the fact that reflexes [were] present and she ha[d] no objective neurologic abnormalities." Ex. 4 at 695. Dr. Lahti further commented that Petitioner's imaging did not support multiple sclerosis or cord impingement-type symptoms, and that her anxiety was a possible explanatory factor. *Id*. at 695–96. He advised Petitioner to follow up with her PCP. *Id*. at 696.

Vaccination and Subsequent Neuropathic Symptoms

Petitioner received a flu vaccine on September 10, 2020, at a Walmart in West Linn, Oregon. Ex. 1. Three days later (September 13, 2020), she went to the PWF ER reporting a continuation of her symptoms from three weeks prior, and that she now maintained had progressed, with increased weakness of her extremities, especially the lower extremities, difficulty walking, and some inability to hold items in her hands. Ex. 3 at 84. She also reported that she had received a flu vaccine in the days before (although she provided an erroneous date). *Id*.; *see also id*. at 105 ("Patient states, I had a flu shot on Friday 9/11/2020 at Walmart in Westlinn [sic], and *the following day (Saturday)* was when I began to have weakness in my legs but more notably in my left leg.") (emphasis added).

Exam revealed decreased strength in Petitioner's bilateral lower extremities, deceased bilateral grip strength, and loss of bilateral patellar reflexes with biceps and brachioradialis reflexes present. Ex. 3 at 87. And her lumbar puncture test results showed an elevated cerebrospinal fluid ("CSF") protein of 69, a white blood cell ("WBC") count of 2, and a red blood cell count of 29. *Id*. at 90. She was diagnosed with GBS and admitted to the hospital to begin a five-day course of IVIG plus daily occupational therapy ("OT"). *Id*. at 121, 128.

Ms. Borgelt began her IVIG course on September 14th. Ex. 3 at 129. In a hospitalist history and physical, she described numbness in her hands and feet for four weeks (hence pre-dating vaccination), but that she started feeling weakness in her arms and legs the day *after* getting her flu vaccine. *Id*. at 112. In an OT evaluation, Petitioner required minimal assistance with her activities of daily living ("ADLs") and used a front-wheeled walker to make transfers. *Id.* at 134. Her back pain was to be managed with medication, although she continued to complain of numbness in her extremities. *Id*. at 143.

Between September 15–18, 2020, Petitioner's numbness and tingling stabilized, and she noticed improved strength. Ex. 3 at 157, 170, 178, 194–203. She thereafter was transferred to Providence Portland Inpatient Rehabilitation, wherein she made significant improvements in her ADLs, sensory deficits, balance, and strength. Ex. 18 at 182–87. Petitioner was discharged on September 26, 2020. *Id.*

Repeated Hospitalizations in the Fall of 2020

The very next day (September 27, 2020), Petitioner returned to the PWF ER, reporting that she developed a patch of numbness in the back of her throat causing problems swallowing, and that she was experiencing increasing pain spreading towards her knees. Ex. 3 at 44. On exam, she was able to swallow without difficulty, but had absent deep tendon reflexes ("DTRs"), general weakness, and strength deficits. *Id*. at 47. The treating hospitalist noted that petitioner had "GBS related to receiving the influenza vaccine," and that she consulted neurology, who advised further IVIG was not recommended at this time, but instead that Petitioner should be discharged home with close outpatient follow-up in clinic. *Id*. at 47–48.

On September 28, 2020, Petitioner went back to the St. Vincent ER, complaining of ongoing neurologic issues (progressive numbness in her face, chest, legs, and buttocks; an inability to walk and lift her arms). Ex. 4 at 15, 24, 39. She was admitted to the hospital for observation and concern for a GBS recurrence. *Id*. at 19–20. While hospitalized, she was evaluated by internist Jasper Erickson, M.D., who reasoned that because she had diffuse weakness, poor effort with lack of contralateral pressure, areflexia, and numbness, the latter of which is "atypical for GBS," an MRI would be diagnostically useful, in order to distinguish between an etiology of metastatic disease vs. transverse myelitis. Ex. 4 at 38. Dr. Erickson also proposed that Petitioner's symptom reoccurrence could "possibly be related to GBS, though the timing appears to be atypical of the usual course," and that "CIDP may be considered at this point due to duration of symptoms from onset." *Id.* The thoracic and lumbar spine MRI showed no abnormal T2 signal or enhancement in Petitioner's cord, no abnormal nodularity or neve root thickening in the cauda equina, and mild degenerative changes without neural encroachment. *Id.* at 50.

Petitioner also received a neurology consultation from Biggya Saptoka, M.D. Dr. Saptoka ordered an electromyography/nerve conduction study[3] ("EMG/NCS") for further evaluation. Ex. 4 at 52. This testing (performed October 1, 2020) showed generalized predominantly distal, motor greater than sensory, demyelinating neuropathy, consistent with GBS. *Id.* at 50. In light of these findings (as well as Petitioner's favorable response to prior IVIG infusions), Dr. Sapkota ordered a second, five-day course of IVIG, as well as physical therapy ("PT") and OT treatment, along with other neuropathic pain medication. *Id.* at 143.

[3] "Electromyography" is defined as "an electrodiagnostic technique for recording the extracellular activity (action potentials and evoked potentials) of skeletal muscles at rest, during voluntary contractions, and during electrical stimulation; performed using any of a variety of surface electrodes, needle electrodes, and devices for amplifying, transmitting, and recording the signals." *Electromyography*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=15854&searchterm=electromyography (last visited Jan. 5, 2026).

"Nerve Conduction Study" is defined as "a diagnostic test that evaluates the function of your peripheral nerves. An NCS can help detect the presence and extent of peripheral nerve damage." *Nerve Conduction Study*, Cleveland Clinic, https://my.clevelandclinic.org/health/treatments/24821-nerve-conduction-study (last visited Jan. 5, 2026).

On October 6, 2020, Petitioner was discharged to inpatient rehabilitation. Ex. 7 at 279. Later that month, she saw neurologist John Zurasky, M.D., who noted improving lower extremity weakness and bilateral hand weakness on exam. Ex. 18 at 148–49. Petitioner was discharged home on October 20, 2020, after having made functional progress. *Id.* at 114. But she went back to the St. Vincent ER again the day after, complaining of chest discomfort, along with continued lower extremity weakness, pain, and difficulty walking that had resulted in a fall. Ex. 5 at 620, 626. On exam, Petitioner displayed intact upper extremity grip strength with intermittent carpal spasm, significant distal lower extremity weakness, and absent bilateral patellar DTRs. *Id*. at 622. Her chest pain was thought to be musculoskeletal in nature, and she was to continue a prior prescription upon discharge home that day. *Id*. at 624.

Later in October 2020, Petitioner had a follow-up visit with Dr. Cruz. Ex. 2 at 73. At this time, she provided a history in which she reported that she had felt tingling of her hands the day *prior* to vaccination, but symptoms of weakness and increased paresthesia, numbness, and tingling occurring a "few days after" vaccination. *Id.* at 74. Petitioner reported receiving home health PT and OT, and was now using a wheelchair. *Id*. Dr. Cruz reconciled Petitioner's medications, to include increasing Petitioner's antidepressant medication dosage. *Id*. at 72–74.

On October 30, 2020 (now more than seven weeks post-vaccination), Petitioner went to the OHSU ER for worsening weakness and increased falls in the prior few days. Ex. 6 at 9. She reported that in the morning, she could not grip her cup and had decreased strength and mobility in her hands, difficulty walking and lower leg sensation, and that she felt an "electricity" running down her neck to her toes when she touched her chin to her chest. *Id*. She also again claimed to have experienced some symptoms within days of vaccination, and that she did not believe her second round of IVIG had been beneficial. *Id.* On exam, Petitioner displayed a cranial nerve deficit, decreased bilateral strength in her upper extremities, an inability to lift her legs while sitting or attempting to ambulate, and absent bilateral lower extremity reflexes. *Id*. at 17.

Ms. Borgelt was again admitted to the hospital, and seen by neurologist Tae Kim, M.D. Ex. 6 at 21. Dr. Kim found her exam notable for distal more than proximal weakness that was greater in her legs than arms, areflexia, and diminished sensation in all modalities in the distal lower extremities. *Id.* at 39. Her labs were also thought notable, revealing for a CSF protein level of 69, WBC count of 2, elevated erythrocyte sedimentation rate, and positive Sjogren's syndrome-related antibodies.[4] *Id*. Although Dr. Kim accepted that Petitioner's *initial* presentation and

[4] Sjogren Syndrome is defined as "a symptom complex of unknown etiology, usually occurring in middle-aged or older women, marked by the triad of keratoconjunctivitis sicca with or without lacrimal gland enlargement, xerostomia with or without salivary gland enlargement, and the presence of a connective tissue disease, usually rheumatoid arthritis but sometimes systemic lupus erythematosus, scleroderma, or polymyositis. An abnormal immune response has been implicated." *Sjogren Syndrome*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=111409 (last visited Jan. 5, 2026).

workup was consistent with GBS, he felt her subsequent worsening was more reflective of early CIDP (although he discounted vasculitis or motor neuron disease). *Id*.

Dr. Kim ordered repeat MRI studies, a repeat lumbar puncture, and additional labs. Ex. 6 at 40. Petitioner's lumbar puncture later that day showed an elevated CSF protein of 117 and an elevated WBC of 7. *Id.* But an October 31, 2020 cervical spine MRI study showed only minor degenerative change, while a brain MRI revealed no concerns. Ex. 6 at 165, 168. At a November 1, 2020 exam with neurologist Michael Lane, M.D., Petitioner was able to lift her legs against resistance, although she continued to have absent reflexes. *Id.* at 65. Per Dr. Lane's recommendation, Petitioner received a maintenance IVIG dose before considering plasmapheresis. *Id*. at 65, 70.

A culture of the CSF performed November 2, 2020, showed no growth, and Petitioner's CSF oligoclonal bands results were negative. Ex. 6 at 106, 108. Petitioner also continued to experience some soreness and stiffness in her legs and hands that was worse following PT sessions, but she reported no new weakness, numbness, or tingling. *Id.* at 85. That same month, Petitioner tested positive for ANA antibodies and some other Sjogren's syndrome-related antibodies, which were deemed possible evidence of a rheumatologic etiology (or a false positive in the setting of IVIG). *Id.* at 22.

Petitioner was discharged from OHSU hospital on November 5, 2020, with a walker and PT/OT services scheduled. Ex. 6 at 25. Her differential diagnosis on discharge included "AIDP non-responsive to IVIg (which means CIDP progression more likely), paraneoplastic or infiltrative neoplastic acute/subacute peripheral neuropathy (given breast cancer history), [or] inflammatory/autoimmune PN [pruigo nodularis]," although "[h]er overall picture, however, was most suggestive of AIDP" as "she [was] not quite at 8 week cut-off for AIDP nadir vs CIDP" and "illness/symptom progression will clarify this picture." *Id.*

For the remainder of the fall of 2020, Petitioner had numerous follow-up visits with her treaters. She saw a different neurologist, Dr. David Wilkinson, on November 12, 2020. Ex. 11 at 308. Her neurologic exam showed improved motor function, especially in her legs, but significant motor limitation persisted that was likely compounded by numbness, fatigue, and pain. *Id.* at 308. Dr. Wilkinson noted the lack of clinical evidence for progressive peripheral neuropathy to support a CIDP diagnosis at that time—although a relapse of symptoms would substantiate that diagnosis. *Id.* She returned to Dr. Wilkinson in mid-November, reporting continued weakness and worsening sensation/pain in her hands. *Id.* at 295. Dr. Wilkinson continued to frame the etiologic explanation for her ongoing condition to reflect either CIDP or persistent GBS weakness, and proposed an empiric trial of plasmapheresis sessions, which occurred in the second half of November. *Id.* at 295, Ex. 4 at 460, 499, 523, 553, 583.

By the time of a December 2, 2020 follow-up, however, Dr. Wilkinson more confidently opined that Petitioner's proper diagnosis was refractory CIDP. Ex. 11 at 269. In addition, it was noted at this time that lab testing Petitioner had undergone the month before now revealed the presence of antibodies to neurofascin-155 (NF-155).[5] *Id.*

Treatment in 2021

On January 11, 2021, Dr. Wilkinson saw Petitioner again and took note of her improvement. Ex. 11 at 238–39. A same-day EMG study showed a generalized sensorimotor polyneuropathy. *Id.* at 234–45. Specifically,

> [t]here appear to be a mixture of axon loss and demyelinating features seen. Demyelination is largely distal with some reduction of proximal conduction velocities. There is also a component of secondary axon loss suspected. Compared with prior nerve conduction study dated 10/1/2020 there is some progression of both components. There is no definite evidence for a focal entrapment neuropathy seen in the setting of the above.

Id. By the end of the month, Petitioner had been discharged from PT (although mainly on account of her immunocompromised status. Ex. 14 at 137.

Petitioner continued to demonstrate improvement in the February–March 2021 timeframe. Ex. 2 at 141–42, 145 (visit with Dr. Cruz); Ex. 11 at 208 (February 17, 2021 follow-up visit with Dr. Wilkinson); Ex. 1 at 197 (March 17, 2021 visit with Dr. Wilkinson). She had another ER visit in late-April, but it related to a distinguishable concern. Ex. 5 at 252, 255 (treatment for embolic phenomenon).

By September 2021, Petitioner reported some ongoing bilateral paresthesias in her lower extremities plus muscle weakness and easy fatiguability. Ex. 12 at 199–200. At another follow-up visit with Dr. Wilkinson that month, Dr. Wilkinson proposed that her ongoing chronic severe neuropathy reflected neurofascin positive refractory CIDP, and that without further therapy, he was concerned she would relapse. Ex. 1 at 155.

On October 25, 2021, Ms. Borgelt underwent a third EMG/NCS, which showed (1) continued evidence for a sensorimotor polyneuropathy reflecting axon loss and significant improvement in diffuse demyelination compared to her January 11, 2021 study; and (2) continued evidence of right median sensory and motor latency prolongation, which may reflect persistent

[5] Neruofascin-155 autoantibodies are understood to be among the most common nodal and paranodal nerve antibodies, and have been associated with CIDP. National Library of Medicine, https://pmc.ncbi.nlm.nih.gov/articles/PMC8673722/ (last visited Jan. 5, 2026).

demyelination from diffuse polyneuropathy, although the response of a superimposed right median neuropathy at the wrist segment could reflect right carpal tunnel syndrome. Ex. 11 at 128–29. Dr. Wilkinson reviewed the results with Petitioner at a telehealth appointment the next day, and proposed more treatments with rituximab,[6] but Petitioner declined in favor of monitoring. *Id.* at 119.

In mid-December 2021, Dr. Wilkinson again saw Petitioner, noting continued improvement except for weakness in her ankle dorsi and plantar flexors. Ex. 11 at 94. Later that day, Petitioner underwent her fourth EMG/NCS, which showed sensorimotor polyneuropathy with axon loss and continued improvement since her prior study; no focal peripheral monotherapy, or carpel tunnel, was evident. *Id.* at 91.

Treatment in 2022 and Beyond

Although Petitioner has filed many additional records reflecting treatment she continued to receive two or more years post-vaccination, most of this evidence bears little on entitlement (although it sadly also reveals Petitioner's struggle in treating breast cancer). She was still experiencing some neuropathic symptoms in April 2022. Ex. 16 at 134. Dr. Wilkinson expressed concerns that relapses were likely if rituximab treatments were ceased. Ex. 11 at 28–29. She reported additional neuropathic weakness in the fall of 2022. Ex. 13 at 236–38. But by February 2023, Dr. Wilkinson proposed that "the CIDP component of her issues [was] not active and [he saw] no indication to consider IVIG or other immunotherapies." Ex. 11 at 17. He did note unchanged neuropathy and some foot pain, which he deemed "quite likely" from her peripheral neuropathy, although plantar fasciitis was "difficult to exclude." *Id*. at 17–18. Petitioner continued to report residual foot neuropathic symptoms in the following month. Ex. 13 at 12.

II. Witness Testimony

A. *Petitioner's Expert – Dr. Zurab Nadareishvili*

Dr. Nadareishvili, a neurologist and stroke specialist, prepared two written reports for Petitioner and testified at hearing. Report, dated May 9, 2024, filed as Ex. 25 (ECF No. 15-4) ("First Nadareishvili Rep."); Report, dated Dec. 3, 2024, filed as Ex. 65 (ECF No. 18-2) ("Second Nadareishvili Rep.").

Dr. Nadareishvili is the Medical Director of the Inpatient Neurology and Comprehensive Stroke Center at VHC Health and a Clinical Professor of Neurology at The George Washington

[6] *Butland v. Sec'y of Health & Hum. Servs.*, No. 07-111V, 2009 WL 1949059, at n.28 (Fed. Cl. Spec. Mstr. June 19, 2009) ("Rituximab (the generic version of Rituxan) is a chimeric, monoclonal antibody against the CD20 antigen, used in the treatment of B-cell non-Hodgkin's lymphoma., B-cell leukemias, and some autoimmune disorders.").

University of School of Medicine and Health Sciences. CV, filed as Ex. 25 (ECF No. 15-4) ("Nadareishvili CV"), at 1. He is licensed in the District of Columbia and Virginia. *Id*. at 2. He holds board certifications in Neurology and Vascular Neurology. *Id*.; Tr. at 6. He treats about twelve GBS and three to five CIDP patients per year, usually referring the latter for subspecialist treatment. First Nadareishvili at 2. His clinical fellowship was in vascular neurology. Tr. at 8. He received his M.D. in 1984, and his Ph.D. in Neurology 1991, from the Tbilisi State Medical Institute in the Republic of Georgia. *Id.* at 7; Nadareishvili CV at 1. Overall, Dr. Nadareishvili's expertise lies more in vascular neurology and stroke treatment than in neuromuscular or peripheral neuropathic disease, or CIDP generally, and he lacks specific experience researching such topics, despite his overall practical familiarity with a variety of neurologic/neuromuscular diseases and conditions. Tr. at 62–64.

Dr. Nadareishvili accepted Petitioner's diagnosis of CIDP, and he agreed that the record supported the conclusion that it had begun prior to her vaccination. Tr. at 14. But he added that antibody testing Petitioner had undergone established that she suffered from a *specific form* of the condition: "NF155," or anti-neurofascin 155 antibody-positive CIDP ("anti-neurofascin CIDP"). Tr. at 13, 14; First Nadareishvili Rep. at 23. He deemed it an uncommon CIDP variant, and that this was the likely form of CIDP Petitioner had been experiencing pre-vaccination—and thus Petitioner likely already possessed the anti-neurofascin antibodies at the time she was vaccinated, even if they were only detected later on. Tr. at 28, 43.

Dr. Nadareishvili stressed that anti-neurofascin CIDP is different in nature and presentation than other forms. One item of literature Dr. Nadareishvili filed on the subject defined anti-neurofascin CIDP to be "a highly specific biomarker of clinically, electrophysiologically, and histopathologically distinct demyelinating polyradiculoneuropathy [in which the] clinical course and outcome are different from typical CIDP, with most patients requiring long-term immune treatment (<2 years) and the utilization of more aggressive immunotherapy." S. Shelly et al., *Neurofascin-155 Immunoglobulin Subtypes*, 97 Neurology e2392 (2021), filed as Ex. 57 (ECF No. 15-36). In particular, anti-neurofascin CIDP featured a "more rapid progression," was more likely to be experienced by younger patients, and "can present with ataxia, tremors, [and] motor deficits." Tr. at 22, 45. It would also feature the kind of symptoms relapse seen with CIDP generally. *Id.* at 66. At the same time, however, little is known about this form of CIDP, Dr. Nadareishvili acknowledged, including what antigen prompts development of the antibody in question. *Id.* at 30–31, 69.

As Dr. Nadareishvili explained, neurofascin-155 is a protein found in the "nodes of Ranvier" along a nerve (gaps in a nerve's outer myelin sheath).[7] *Id.* at 20. This would be the

[7] "Nodes of Ranvier" is defined as "constrictions occurring on myelinated nerve fibers at regular intervals of about 1 mm; at these sites the myelin sheath is absent and the axon is enclosed only by Schwann cell processes." *Nodes of Ranvier*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=93095 (last visited Jan. 5, 2026).

location where antibodies specific to this protein would attack. The neurofascin-155 protein serves as an "adhesion molecule" that is important to nerve cell development, axon targeting, and synapse formation. *Id.* at 24. In addition, Dr. Nadareishvili noted that the anti-neurofascin antibodies in question were *IgG4* antibodies—a particular kind of immune cell that suppresses, rather than promotes, inflammation, but in so doing "blocks other processes," leading to harm. Tr. at 20, 23; First Nadareishvili Rep. at 23–24; J. Kira, *Anti-Neurofascin 155 Antibody-Positive Chronic Inflammatory Demyelinating Polyneuropathy/Combined Central and Peripheral Demyelination: Strategies for Diagnosis and Treatment Based on the Disease Mechanism*, 12 Frontiers in Neurology 1 (2021), filed ad Ex. 42 (ECF No. 15-21). Thus, an autoimmune attack by these autoantibodies against the neurofascin protein would damage nerves. (But this contention flies in the face of Dr. Nadareishvili's statement that anti-neurofascin CIDP "is not classified as IgG4 related disease." Second Nadareishvili Rep. at 4).

Dr. Nadareishvili also noted that the pathologic process associated with anti-neurofascin CIDP was likely mediated in part by a kind of specific T-helper cell (Th2 cells)[8] that promotes the production of anti-inflammatory cytokines, and/or stimulates B cells responsible for production of the relevant IgG4 anti-neurofascin 155 antibodies. Tr. at 20–21, 39–40. (He maintained, however, that some inflammation was still present. *Id.* at 22). (Dr. Nadareishvili tried to draw a parallel between this kind of antibody and T helper cell-driven reaction to an allergic reaction). He also observed that Petitioner had reported some kind of dangerous allergic reaction in the summer of 2020 to shellfish, stressing that this too was understood to be "associated with IgG4 response," and/or a Th2-mediated process, and thus it was possible her earlier reaction was evidence of how her vaccine-associated worsening might have occurred. Tr. at 28–29, 91; Second Nadareishvili Rep. at 4, 10. This could possibly explain why Petitioner's reaction to vaccination was so sudden (even though Dr. Nadareishvili was not otherwise contending the flu vaccine had initiated Petitioner's anti-neurofascin CIDP). Tr. at 30.

The flu vaccine, Dr. Nadareishvili maintained, could cause worsening of the form of CIDP Petitioner experienced. Tr. at 33–34. That vaccine is already known to be capable of causing autoimmune diseases like GBS, and Dr. Nadareishvili cited literature (in particular, case reports) suggesting infections or vaccinations were often associated with CIDP specifically. A. de Souza et al., *Inflammatory Demyelinating Polyneuropathy after the ChAdOx1 nCoV-19 Vaccine may Follow a Chronic Course*, 436 J. Neurological Sciences 1 (2022), filed as Ex. 58 (ECF No. 15-37); J. Brostoff et al., *Post-Influenza Vaccine Chronic Inflammatory Demyelinating Polyneuropathy*, 37 Age and Ageing 229 (2008); Tr. at 36–38. Yet Dr. Nadareishvili admitted that the anti-neurofascin CIDP Petitioner suffered from would not propagate in the same manner, i.e.

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[8] The T-helper cell encourages the production of antibodies to antigens that interact with B-cells. *Helper Cells*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=64157&searchterm=helper+cells (last visited Jan. 5, 2026).

due to molecular mimicry between vaccine antigens and self-nerve protein components, with the production of pathogenic antibodies the end result. Tr. at 18–19, 34–35 (maintaining that the causal theory in this case did not rely on establishing mimicry between flu vaccine protein components and self-antigens), 91.[9] He later observed studies suggesting that a pediatric population developed IgG4 antibodies after receipt of an acellular pertussis-containing vaccine, allowing for the possibility that vaccines might encourage the generation of the anti-neurofascin antibody. *Id.* at 31–32; S. Van der Lee et al., *Whole-cell or Acellular Pertussis Vaccination in Infancy Determines IgG Subclass Profiles to DTaP Booster Vaccination*, 36 Vaccine 220 (2018), filed as Ex. 72 (ECF No. 18-9).

In this case, the flu vaccine likely did cause worsening of Petitioner's symptoms, in Dr. Nadareishvili's view, although he was somewhat uncertain as to *how* this occurred. He maintained that vaccination could promote initial production of proinflammatory cytokines (via Th1 T-helper cells), thereby activating the immune system. Tr. at 32–33. Exacerbation of existing anti-neurofascin CIDP was also possible through post-vaccination stimulation of T and B cells already involved in the pathologic process (via Th2 T-helper cells). Tr. at 42, 46; L. Zhang et al., *Anti-Neurofascin-155 Antibody Mediated a Distinct Phenotype of Chronic Inflammatory Demyelinating Polyradiculoneuropathy*, 271 J. Neurology 4991 (2024), filed a Ex. 73 (ECF No. 18-10). Noting evidence of Petitioner's summer 2020 shellfish allergic reaction, he opined that the vaccine might have constituted some kind of "repetitive stimuli, basically driving worsening in what was first stimuli"—specifically by inducing some kind of IgG4 response post-vaccination, or through promotion of T-helper cells, which would in turn encourage B cells to produce more of the anti-neurofascin 155 antibodies. Tr. at 30; Second Nadareishvili Rep. at 10.

Ultimately, Dr. Nadareishvili admitted that there was not a great deal of evidence speaking to the possibility of worsening due to vaccination, but he contended that the issue was very difficult to research (explaining the paucity of relevant research). Tr. at 43–44, 83–84. He also allowed that his theory "worked" based upon any immunologic stimulus received once a person was already experiencing anti-neurofascin CIDP (and thus it almost did not matter what the cause of that stimulus was). *Id.* at 76. The one-time impact of a vaccine could be enough to explain all that followed later (although Petitioner in this case had experienced multiple relapses). *Id.* at 76, 82–83, 84 (saying "[p]otentially" under his theory, any vaccine received at the time Petitioner was administered the flu vaccine could have caused her exacerbation of symptoms).[10]

[9] Dr. Nadareishvili comparably admitted he had offered no evidence establishing that flu vaccine components had any homology at all with neurofascin 155 protein sequences. Tr. at 69, 70, 75. He also noted he had no direct proof establishing the flu vaccine could spark renewed or additional production of the anti-neurofascin antibodies. *Id.* at 69–70.

[10] Respondent noted on cross-examination, however, that Petitioner had experienced an upper respiratory infection in September 2021 (a year after vaccination), and yet did not appear from the medical records to have suffered a CIDP relapse (thus undermining the contention that general immune stimulation was sufficient to spark worsening). Tr. at 84.

Dr. Nadareishvili stressed that the record showed no other explanation for why Petitioner experienced worsening of symptoms after vaccination. Tr. at 47. And the medical records established that Petitioner's treaters were aware of her vaccination as a possible explanation (although he admitted they may have contemplated a vaccine association based on a preliminary view that Petitioner was experiencing GBS). *Id.* at 49–50, 51–55. He admitted to giving the precise timing of Petitioner's vaccination, compared to when she began to see more severe symptoms, a great deal of weight in assessing the vaccine as causal. *Id.* at 85, 90.

Yet in other parts of his testimony, Dr. Nadareishvili was hard-pressed not to admit the many missing links in his theory. *See, e.g.*, Tr. at 33 ("we don't know whether the vaccine may play a role in this situation"), 60 ("science doesn't know exact mechanism yet or exact antigen or what was driving all this"). He also acknowledged his reliance on the known fact of vaccination as proof of causation. *Id.* at 59 ("there is no other evidence other than this vaccination, basically. . . . this is a clearcut, you know, triggering time point"). But he invoked the difficulty in testing for triggers generally as an explanation for such causal holes. *Id.* at 59–60.

With respect to disease course, Dr. Nadareishvili maintained that Petitioner's CIDP symptoms demonstrably worsened not long after vaccination. Tr. at 15–16. By the time of her first ER visit three days post-vaccination (September 13, 2020), Petitioner was demonstrating more symptoms than she had in August, including new ones like loss of reflexes, and CSF testing revealed elevated protein levels consistent with neuropathic inflammation. *Id.* Thereafter, "she never improved completely" in comparison to her baseline health before August. *Id.* at 17.

Dr. Nadareishvili also addressed the issue of onset of worsening and its relationship to *when* Ms. Borgelt received the flu vaccine. He maintained that at the outset, it would likely take four to six weeks for the anti-neurofascin 155 antibodies to cause the harm that would manifest as first clinical symptoms. Tr. at 44–45. But stimulation of immune memory sufficient to produce *more* of this kind of antibody could occur far more rapidly—within the space of seven days. *Id.* at 45. Here, an immediate exacerbation of Petitioner's condition the day of vaccination was not likely, but days later was reasonable. *Id.* (On cross-examination, however, Dr. Nadareishvili admitted that his initial report seemed to place onset of worsening as the same day as vaccination—something he now seemed to see as unlikely). *Id.* at 89; First Nadareishvili Rep. at 25.

On cross-examination, Dr. Nadareishvili admitted that some literature he had offered to show vaccination-induced worsening of CIDP relied on patient-reported data obtained from questionnaires rather than experimentally-derived data. Tr. at 77–79 (discussing J. Pritchard et al., *Risk of Relapse of Guillain-Barré syndrome or Chronic Inflammatory Demyelinating Polyradiculoneuropathy following Immunisation*, 73 J. Neurol. Neurosurg. Psych. 343, 348 (2002), filed as Ex. 51 (ECF No. 15-30)).

B. *Respondent's Expert – Dr. Marcello Matiello*

Dr. Mattielo, a clinical and academic neurologist, testified for Respondent and authored one written report. Report, dated Aug. 13, 2024 (ECF No. 16-1) ("Matiello Rep.").

Dr. Matiello received his M.D. from the Federal University of Rio de Janeiro in 2000, and M.Sc. from the Mayo Graduate School of Medicine in 2012, among many other fellowships and residencies from 2001–18. Curriculum Vitae, filed a Ex. B (ECF No. 16-19) at 1. He is a board-certified neurologist at Massachusetts General Hospital and an Associate Professor at Harvard Medical School. Matiello Rep. at 2; Tr. at 93. He actively treats a variety of demyelinating central and peripheral nervous system diseases in inpatient and outpatient contexts. In an inpatient setting, Dr. Matiello's service is "primarily dedicated for patients with either neuroimmunology diseases such as GB[S] [sic], CIDP, myasthenia gravis, NMO, MS, … or patients with neurodegeneration.". Tr. at 95; Matiello Rep. at 2. In a clinical setting, Dr. Matiello stated that he has evaluated and treated approximately twenty patients with CIDP in the last ten years. Tr. at 96. He has spent his career focused on investigating clinical and biological susceptibility factors in inflammatory and demyelinating diseases. *Id.* He researches mechanisms of autoimmunity, clinical presentations, and disease progression of diverse neurologic diseases. *Id.* Dr. Mattiello has also contributed to over 50 peer-reviewed papers, predominantly as a first or senior author, and authored 11 book chapters on neurology and neuroimmunology. *Id.*

Dr. Matiello began his testimony with a discussion of anti-neurofascin CIDP (a diagnosis he deemed fit the Petitioner's circumstances). Tr. at 104, 113. He identified neurofascin 155 as "a molecule that is expressed by the Schwann cell[11] which connects with two different proteins in the axon, . . . and this protein anchors the Schwann cell myelin to the axon," allowing the Schwann cell to perform its function of myelin production, *Id.* at 104–05. Antibodies to neurofascin 155 constitute a recent discovery, Dr. Matiello contended, and when generated they can bind to neurofascin 155 and "cause disruption" of the connection, harming the nerve and impacting its performance. *Id.* at 106, 107, 111. He considered this form of CIDP to be particularly rare, although he felt much was already known about it. *Id.* at 123–24.

The antibodies in question, Dr. Matiello stated, are of an "IgG4 subclass" that performs differently from other kinds of antibodies. Tr. at 107. In particular, he observed that the anti-neurofascin antibody "doesn't have any complement-mediated destruction,"[12] and "it does not have this ability to bring more inflammation or immune response." *Id.* at 107, 108, 122–23; J. Ng et al., *Neurofascin as a Target for Autoantibodies in Peripheral Neuropathies*, 79 Neurology 2241

[11] The Schwann cell is a type of glial cell (immune cells that provide structure for neurons) in the peripheral nervous system that insulates and supports nerve fibers. *Schwann Cell*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=64407&searchterm=Schwann+cell (last visited Jan. 5, 2026).

[12] Complement is a component of the innate immune system that defends against foreign pathogens.

(2012), filed as Ex. A-7 (ECF No. 16-8); H. Koike et al., *Paranodal Dissection in Chronic Inflammatory Demyelinating Polyneuropathy with Anti-Neurofascin-155 and Anti-Contactin-1 Antibodies*, 88 J. Neurol. Neurosurg. Psych. 465, 472 (2017), filed as Ex. A-13 (ECF No. 16-14). Dr. Matiello later emphasized that "the evidence in this disease is that the disruption is because of a structural damage," and not due to an overactive inflammatory environment. Tr. at 118. At the same time, however, Dr. Matiello agreed with Dr. Nadareishvili that some proinflammatory T-helper cells were involved in the disease process. *Id.* at 124–26. He also proposed, consistent with Dr. Nadareishvili, that Petitioner likely possessed these anti-neurofascin antibodies prior to vaccination (although he could not say what had caused them to appear). *Id.* at 129, 152, 157.

Thus, anti-neurofascin CIDP stands somewhat alone as a "nodopathy" (since the neurofascin 155 protein is found in the Nodes of Ranvier location on the nerve surface) that usually impacts younger patients (up to age 40), with more rapid presentation, and faster course to nadir as well. Tr. at 108, 109. It more often features tremors or ataxia in comparison to typical cases of CIDP (although all involve symptoms relapses), and IVIg (a standard treatment for other peripheral neuropathies like GBS) does not work as well—and it did not for Petitioner either. *Id.* at 10–11. Its rapid/acute appearance, however, can cause treaters to initially confuse it with GBS (as it appeared to occur in this case). *Id.* at 109. Dr. Matiello firmly denied that this form of CIDP has any known antigenic trigger. *Id.* at 127, 165.

While Dr. Matiello could not firmly state what might possibly drive initial production of the anti-neurofascin 155 antibodies, he observed what science does know about IgG4 antibody production. In particular, certain items of literature suggest (in keeping with their parallel involvement in allergic reactions) that the IgG4 antibody is produced in reaction to repeated/chronic exposure to the same allergens—perhaps reflective of an immune system "antiallergy type of response" that aims to be "protective against that specific allergen." Tr. at 119, 128; T. Rispens & M. Huijbers, *The Unique Properties of IgG4 and its Roles in Health and Disease*, Nature Rev. Immunology (2023), filed as Ex. A-11 (ECF No. 16-12). Dr. Matiello contrasted such a scenario with the one-time receipt of a flu vaccine (or even its annual administration). Tr. at 120. There was, he maintained, no evidence in this case that Petitioner had experienced repeated exposure to the same antigen sufficient to generate an IgG4 production response. *Id.* at 129.

Dr. Matiello also contested Dr. Nadareishvili's argument that Petitioner's shellfish allergic reaction was relevant to causation, arguing that for an IgG4 response to occur, a person would need to be "eating a ton of shellfish," and not just have had one prior exposure to such an allergen (and thus he did not deem that earlier occurrence to have had the capacity to initiate antibodies cross-reactive with neurofascin 155). *Id.* at 120, 121, 127 ("I'm not familiar with the claim that shellfish allergy—[that] people with that allergy may develop severe reactions to vaccination" as well). Allergic reactions could not be deemed comparable to an autoimmune response. *Id.* at 128.

The flu vaccine, Dr. Matiello maintained, had not likely had anything to do with Petitioner's CIDP course. He indicated no awareness of independent medical or scientific literature linking the two. Tr. at 130, 136. Treaters may have initially deemed an association possible, but they did so based on her first presentation—which looked like GBS (a neuropathy known to be vaccine-associated—but distinguishable from CIDP). *Id.* at 133–34, 162. Later on, treaters did not propose that association. *Id.* at 134–35.

Dr. Matiello further rejected Petitioner's arguments about how her CIDP course purportedly worsened post-vaccination (although he did seem to accept it *had* worsened overall). Tr. at 137, 161 ("I would say it's acutely worsening of something that is already presenting subacutely"). Dr. Nadareishvili had not demonstrated, Dr. Matiello contended, that an immune stimulus of the kind associated with a vaccination was necessary to cause a CIDP flare or relapse. *Id.* at 130. Even when Petitioner later had experienced immune stimulation (for example, after receipt of a COVID-19 vaccine in September 2021 and February 2022, or a viral infection in September 2021), she had not also suffered any CIDP symptoms relapses. *Id.* at 143–45.[13]

Although Dr. Nadareishvili had opined that Petitioner never "returned to baseline" after receipt of the flu vaccine, Dr. Matiello felt that "after the damage [from CIDP] happens, it's there" at the nerve nodes, producing persistent disability. *Id.* at 113. He also proposed that the delay in diagnosis (along with the later substitution of Rituximab for IVIg) may have prolonged the damage's extent. *Id.* at 114. In addition, the record did show Petitioner had experienced some relapses in the fall of 2020, but he deemed this to be consistent with CIDP generally. *Id.* at 126–27. They could not credibly be associated with a one-time vaccination. Tr. at 138–40. And literature that purportedly addressed such circumstances was not specific enough to the anti-neurofascin CIDP variant Petitioner had experienced. *Id.* at 140–42; K. Kuitwaard et al., *Recurrences, Vaccinations and Long-term Symptoms in GBS and CIDP*, 14 J. Peripheral Nervous System 310, 313 (2009), filed as Ex. 43 (ECF No. 15-22).

With respect to onset, Dr. Matiello deemed Petitioner to have first experienced post-vaccination symptoms the same day as the September 10, 2024 vaccination, since she sought hospital care three days later (but at that time complained of symptoms that she reported began three days earlier). Tr. at 115–16. He also noted that Dr. Nadareishvili seemed to accept the same onset. *Id.* at 126 (referencing Second Nadareishvili Rep. at 2).

Such a fast onset of worsening was not immunologically plausible, Dr. Matiello contended. Tr. at 116. Under Dr. Nadareishvili's theory, the immune system would first need to "recognize"

[13] On cross-examination, Dr. Matiello admitted that an infection nevertheless posed dangers for someone with a neuropathic illness. Tr. at 171–72. But he emphasized that this was "not because the infection itself would produce more immunogenesis, and destruction of the nerve," but instead because of its capacity to create "a byproduct of being febrile or not feeling super well." *Id.* at 171. And he distinguished between the immunologic impact of a vaccine generally from a wild infection. *Id.* at 173.

contents of the flu vaccine, present them to the Th2 cells, and then the latter go on to impact existing B cell production of the anti-neurofascin IgG4 antibodies. *Id.* at 116, 124–25. That multi-step process could take "anywhere from 14 days to entire months or a month and a half." *Id.* at 117, 121, 123 ("IgG4 takes a long time" to harm neuronal nerves sufficient to result in CIDP symptoms), 130–31; Ng at 2246 Fig. 4. Even if the immune system's "memory" of prior exposure to flu vaccine antigens caused a shorter initial response, that would still take two to four days at a minimum. *Id.* Thus, the actual timeline of Petitioner's CIDP worsening was too fast to be consistent with the proposed vaccine-induced pathology. *Id.* at 118. And Dr. Nadareishvili seemed to place great weight on the mere temporal association between vaccination and worsening. *Id.* at 131.

III. Procedural History

The matter was initiated in July 2023, and activated out of "pre-assignment review" in October of that year. After Respondent's Rule 4(c) Report set forth his objection to entitlement in January 2024, the parties began the process of obtaining and filing reports for the experts discussed above. The case went to hearing in May 2025, and is now ripe for resolution.

IV. Applicable Legal Standards

A. *Petitioner's Overall Burden in Vaccine Program Cases*

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a "Table Injury"—i.e., an injury falling within the Vaccine Injury Table—corresponding to one of the vaccinations in question within a statutorily prescribed period of time or, in the alternative, (2) that his illnesses were actually caused by a vaccine (a "Non-Table Injury"). *See* Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); *see also Moberly ex rel. Moberly v. Sec'y of Health & Hum. Servs.,* 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006).[14] There is no Table claim for CIDP caused or aggravated by the flu vaccine.

For both Table and Non-Table claims, Vaccine Program petitioners bear a "preponderance of the evidence" burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact's existence." *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enter. v. United States*, 6 Cl. Ct.

[14] Decisions of special masters (some of which I reference in this ruling) constitute persuasive but not binding authority. *Hanlon v. Sec'y of Health & Hum. Servs.*, 40 Fed. Cl. 625, 630 (1998). By contrast, Federal Circuit rulings concerning legal issues are binding on special masters. *Guillory v. Sec'y of Health & Hum. Servs.*, 59 Fed. Cl. 121, 124 (2003), *aff'd* 104 F. Appx. 712 (Fed. Cir. 2004); *see also Spooner v. Sec'y of Health & Hum. Servs.*, No. 13-159V, 2014 WL 504728, at *7 n.12 (Fed. Cl. Spec. Mstr. Jan. 16, 2014).

476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y Health & Hum. Servs*., 165 F.3d 1344, 1352–53 (Fed.Cir.1999)); *Pafford v. Sec'y of Health & Hum. Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen*, 418 F.3d at 1278: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury."

Each of the *Althen* prongs requires a different showing. Under *Althen* prong one, petitioners must provide a "reputable medical theory," demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355–56 (citations omitted). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 549.

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or even a generally accepted medical theory. *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1378–79 (Fed.Cir.2009) (citing *Capizzano*, 440 F.3d at 1325–26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras v. Sec'y of Health & Hum. Servs*, 121 Fed. Cl. 230, 245 (2015), *vacated and remanded*, 844 F.3d 1363 (Fed. Cir. 2017).

In discussing the evidentiary standard applicable to the first *Althen* prong, the Federal Circuit has consistently rejected the contention that it can be satisfied merely by establishing the proposed causal theory's scientific or medical *plausibility*. *See Cerrone v. Sec'y of Health & Hum. Servs.*, 146 F.4th 1113, 1122 (Fed. Cir. 2025); *Kalajdzic v. Sec'y of Health & Hum. Servs.*, No.

2023-1321, 2024 WL 3064398, at *2 (Fed. Cir. June 20, 2024) (arguments "for a less than preponderance standard" deemed "plainly inconsistent with our precedent" (*citing Moberly*, 592 F.3d at 1322)); *Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019); *see also Demore v. Sec'y of Health & Hum. Servs.*, No. 20-1265V, 2024 WL 4542934 (Fed. Cl. Spec. Mstr. Sept. 26, 2024), *aff'd*, No. 20-1265V, 2025 WL 868902, at *4 (Fed. Cl. Mar. 20, 2025) (rejecting the argument that a petitioner's burden is to prove that a causation theory is *plausible* and instead requiring petitioner to prove the theory by a preponderance of the evidence) (emphasis added). And petitioners always have the ultimate burden of establishing their *overall* Vaccine Act claim with preponderant evidence. *W.C. v. Sec'y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted); *Tarsell v. United States*, 133 Fed. Cl. 782, 793 (2017) (noting that *Moberly* "addresses the petitioner's overall burden of proving causation-in-fact under the Vaccine Act" by a preponderance standard).

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375–77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine "did cause" injury, the opinions and views of the injured party's treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 ("medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury'") (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras*, 993 F.2d at 1528.

Medical records and statements of a treating physician, however, do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Hum. Servs.*, 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Hum. Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec'y of Dept. of Health & Hum. Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den'd*, 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 F. Appx. 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *de Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must align with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Hum. Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. den'd after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 503 F. Appx. 952 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Hum. Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for rev. den'd* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. *Legal Standards for Significant Aggravation Claim*

Where a petitioner alleges significant aggravation of a preexisting condition, the *Althen* test is expanded, and the petitioner has additional evidentiary burdens to satisfy. *Loving v. Sec'y of Health & Hum. Servs*., 86 Fed. Cl. 135, 144 (2009). In *Loving*, the Court of Federal Claims combined the *Althen* test with the test from *Whitecotton v. Sec'y of Health & Hum. Servs.*, 81 F.3d 1099, 1107 (Fed. Cir. 1996), which related to on-Table significant aggravation cases. The resultant "significant aggravation" test has six components, which require establishing:

> (1) the person's condition prior to administration of the vaccine, (2) the person's current condition (or the condition following the vaccination if that is also pertinent), (3) whether the person's current condition constitutes a 'significant aggravation' of the person's condition prior to vaccination, (4) a medical theory causally connecting such a significantly worsened condition to the vaccination, (5) a logical sequence of cause and effect showing that the vaccination was the reason for the significant aggravation, and (6) a showing of a proximate temporal relationship between the vaccination and the significant aggravation.

Loving, 86 Fed. Cl. at 144; *see also W.C.*, 704 F.3d at 1357 (holding that "the *Loving* case provides the correct framework for evaluating off-table significant aggravation claims"). In effect, the last three prongs of the *Loving* test correspond to the three *Althen* prongs.

In *Sharpe v. Sec'y of Health & Hum. Servs.,* 964 F.3d 1072 (Fed. Cir. 2020), the Federal Circuit further elaborated on the *Loving* framework. Under Prong (3) of the *Loving* test, A Petitioner need not demonstrate an *expected* outcome, but merely that her current-post vaccination condition was worse than pre-vaccination. *Sharpe*, 964 F.3d at 1081. And a claimant may make

out a prima facie case of significant aggravation overall without eliminating a preexisting condition as the potential cause of her significantly aggravated injury (although the Circuit's recasting of the significant aggravation standard still permits Respondent to attempt to establish alternative cause, after the burden of proof has shifted to Respondent). *Id.* at 1083.

It remains uncertain as to what *degree* of aggravation of a pre-vaccination condition would be enough to be considered "significant." The Circuit has made clear that the level of aggravation need not be shown to be catastrophic. *Osenbach v. Sec'y of Health & Hum. Servs.*, No. 2024-1663, 2025 WL 2387944, at *4 (Fed. Cir. Aug. 18, 2025) ("[r]equiring a petitioner to demonstrate that vaccination "wholly altered" their pre-vaccination condition would be at odds with our holding in *Sharpe v. Sec'y of Health & Hum. Servs*. that the *Loving* test does not require petitioners to provide conclusive evidence linking their vaccines to their significant aggravation"). But this aspect of *Osenbach* (which otherwise affirmed dismissal of a significant aggravation claim) seems to pertain only to the well-understood fact that worsening need only be demonstrated *preponderantly* – like any other *Loving* or *Althen* prong.

At a minimum, then, a claimant's injury or disease course must *likely* exceed what that individual might have experienced but for vaccination. A transient worsening (say, due to usual vaccine-associated malaise, experienced immediately after vaccination but which subsides thereafter) is not sufficient—in keeping with the fact that all Program claimants must meet the six-month "severity requirement."[15]

C. *Legal Standards Governing Factual Determinations*

The process for making determinations in Vaccine Program cases regarding factual issues begins with consideration of the medical records. Section 11(c)(2). The special master is required to consider "all [ ] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as the "results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." Section 13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec'y of Health & Hum. Servs*., 3 F.3d 415, 417 (Fed. Cir.

[15] A person with an existing illness could not rely on the mere fact of that illness and its course to prove severity, since it is likely the individual could expect the illness to persist *independent* of vaccination. Rather, it is reasonable to require they preponderantly establish that any symptoms or disease worsening that can be linked to the vaccine results in more than six months of sequelae *specific* to the vaccine's impact. A transient post-vaccination reaction would not meet this standard, no matter how individually severe, if the reaction subsided in less than the Act's six-month timeframe. *See Faoro v. Sec'y of Health & Hum. Servs.*, No. 10-704V, 2016 WL 675491, at *27 (Fed. Cl. Spec. Mstr. Jan. 29, 2026), *mot. for review den'd*, 128 Fed. Cl. 61 (Fed. Cl. Apr. 11, 2016) (finding that "the vaccinations would not have changed [petitioner's] clinical course and thus, the vaccinations did not significantly aggravate her preexisting condition").

1993) (determining that it is within the special master's discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such determination is evidenced by a rational determination).

As noted by the Federal Circuit, "[m]edical records, in general, warrant consideration as trustworthy evidence." *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Hum. Servs.*, 95 Fed. Cl. 598, 608 (2010) ("[g]iven the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law"), *aff'd*, *Rickett v. Sec'y of Health & Hum. Servs*., 468 F. App'x 952 (Fed. Cir. 2011) (non-precedential opinion). A series of linked propositions explains why such records deserve some weight: (i) sick people visit medical professionals; (ii) sick people attempt to honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 11–685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec'y of Health & Hum. Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993 F.2d at 1525 (Fed. Cir. 1993) ("[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms").

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Hum. Servs*., No. 03–1585V, 2005 WL 6117475, at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are often found to be deserving of greater evidentiary weight than oral testimony—especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also Murphy v. Sec'y of Health & Hum. Servs*., 23 Cl. Ct. 726, 733 (1991), *aff'd per curiam*, 968 F.2d 1226 (Fed. Cir. 1992), *cert. den'd*, *Murphy v. Sullivan*, 506 U.S. 974 (1992) (citing *United States v. United States Gypsum Co.*, 333 U.S. 364, 396 (1947) ("[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.")).

However, the Federal Circuit has also noted that there is no formal "presumption" that records are accurate or superior on their face to other forms of evidence. *Kirby v. Sec'y of Health & Hum. Servs.,* 997 F.3d 1378, 1383 (Fed. Cir. 2021). There are certainly situations in which compelling oral or written testimony (provided in the form of an affidavit or declaration) may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec'y of Health & Hum. Servs*., 69 Fed. Cl. 775, 779 (2006) ("like any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking"); *Lowrie*, 2005 WL 6117475, at *19 ("[w]ritten records which are, themselves, inconsistent, should be accorded less

deference than those which are internally consistent") (quoting *Murphy*, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Hum. Servs*., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be "consistent, clear, cogent, and compelling." *Sanchez*, 2013 WL 1880825, at *3 (citing *Blutstein v. Sec'y of Health & Hum. Servs*., No. 90–2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *La Londe v. Sec'y of Health & Hum. Servs*., 110 Fed. Cl. 184, 203–04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

D. *Analysis of Expert Testimony*

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec'y of Health & Hum. Servs*., 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594–96 (1993). *See Cedillo v. Sec'y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999). Under *Daubert*, the factors for analyzing the reliability of testimony are:

> (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.

Terran, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592–95).

In the Vaccine Program the *Daubert* factors play a slightly different role than they do when applied in other federal judicial settings, like the district courts. Typically, *Daubert* factors are

employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable or could confuse a jury. By contrast, in Vaccine Program cases these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Hum. Servs*., 94 Fed. Cl. 53, 66–67 (2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted"). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. *See*, *e.g.*, *Snyder*, 88 Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts in order to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Hum. Servs*., 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 146 (1997)); *see also Isaac v. Sec'y of Health & Hum. Servs*., No. 08–601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for review den'd*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. App'x 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Hum. Servs*., 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act").

E. *Consideration of Medical Literature*

Both parties filed numerous items of medical and scientific literature in this case, but not all such items factor into the outcome of this case. While I have reviewed all the medical literature submitted in this case, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner's case—just as I have not exhaustively discussed every individual medical record filed. *Moriarty v. Sec'y of Health & Hum. Servs.*, No. 2015–5072, 2016 WL 1358616, at *5 (Fed. Cir. Apr. 6, 2016) ("[w]e generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision") (citation omitted); *see also Paterek v. Sec'y of Health & Hum. Servs.,* 527 F. App'x

875, 884 (Fed. Cir. 2013) ("[f]inding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered").

ANALYSIS

I. Overview of CIDP and its Treatment in Program Cases

CIDP generally is defined as "a slowly progressive, autoimmune type of demyelinating polyneuropathy characterized by progressive weakness and impaired sensory function in the limbs and enlargement of the peripheral nerves." *CIDP*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=99346&searchterm=chronic+inflammatory+demyelinating+polyneuropathy (last visited Jan. 2, 2026). Both experts agree that Petitioner was properly diagnosed with anti-neurofascin CIDP (a CIDP variant), based on antibody testing results, as well as Petitioner's course and symptoms. Tr. at 13, 157. They also accept that anti-neurofascin CIDP (a) is mediated by a specific kind of IgG4 antibody, and (b) results initially in an acute presentation inconsistent with other forms of CIDP. *Id.* at 19, 107, 108–09. Anti-neurofascin CIDP is less characterized by excessive inflammation and the damage that comes with it than by structural harm to nerve cells found around myelin nodes (and therefore treatments like IVIg so common to other peripheral neuropathies are less successful). *Id.* at 110, 11. And it is not disputed that Petitioner's anti-neurofascin CIDP likely began *prior* to her receipt of the flu vaccine.

CIDP is often compared to GBS—another kind of inflammatory and demyelinating polyneuropathy characterized by an acute onset, rapid progression, symmetric muscle weakness and hyporeflexia or areflexia. *See DeVaughn v. Sec'y of Health & Hum. Servs.*, No. 22-832V, 2025 WL 758128 (Fed. Cl. Spec. Mstr. Feb. 10, 2025) (discussing the common overlap found in GBS and CIDP). But the conditions can be distinguished, and should be. GBS is monophasic and unresponsive to steroid treatment (unlike CIDP). *Nieves v. Sec'y of Health & Hum. Servs.*, No. 18-1602V, 2023 WL 3580148, at *35 (Fed. Cl. Spec. Mstr. May 22, 2023), *mot. for review den'd*, 167 Fed. Cl. 422 (2023). In addition, GBS and CIDP are not understood to have the same self-targets for cross-reactive attack. *Mason v. Sec'y of Health & Hum. Servs.*, No. 17-1383V, 2022 WL 600415, at *26 (Fed. Cl. Spec. Mstr. Feb. 4, 2022).

Despite their differences, it is not uncommon for CIDP to present *initially* with symptoms comparable to what is featured in GBS—even acutely—and this is especially so for the CIDP variant at issue. Treaters thus often assume they are "seeing" GBS upon first examination and testing of a patient. Because of this overlap, Program petitioners may characterize CIDP as merely "long GBS." *See*, *e.g.*, *Nieves*, 2023 WL 3580148, at *35. The temptation to avoid distinguishing between the two diseases likely stems from the fact that GBS has been credibly linked to certain

vaccinations (namely the influenza vaccine),[16] while the evidence supporting a causal link between CIDP and vaccines is less robust. *Id.* at *35 ("[E]vidence that strongly supports a GBS-flu vaccine causal relationship rings weaker when applied to CIDP"). But it remains the case that CIDP and GBS are separate, immune-mediated polyneuropathies, even if they share many features. *See*, *e.g., Houston v. Sec'y of Health & Hum. Servs.*, No. 18-420V, 2021 WL 4259012, at *17 (Fed. Cl. Spec. Mstr. Aug. 19, 2021) (noting CIDP vs. GBS distinctions).

It is thus is *improper* when deciding a Vaccine Act claim to treat CIDP and GBS as interchangeable.[17] *See Nieves*, 2023 WL 3580148 at *36 ("[T]he overlap between GBS and CIDP cannot be employed as a shortcut to entitlement…"); *see also Howard*, 2022 WL 4869354 at *22 ("[F]or purposes of Program determinations, it is improper to think of GBS and CIDP as 'two sides of the same coin,' despite their overlap…Petitioners cannot just 'borrow' what is known about GBS and vaccination generally as a template for proving causation in the context of a CIDP injury"). I will therefore not blindly apply the evidence supporting a causal link between GBS and other vaccines to this case. While *some* aspects of what is understood about GBS and its relationship to vaccines may well be relevant to my analysis, Petitioner's success ultimately turns on whether she has preponderantly linked *CIDP* to the flu vaccine.

Nevertheless, many Program decisions have assumed that the medical and scientific evidence supporting a GBS-flu vaccine link applies with the same force to CIDP. *See*, *e.g.*, *Jastisan v. Sec'y of Health & Hum. Servs.*, No. 13-937V, 2016 WL 4761950 (Fed. Cl. Spec. Mstr. Aug. 10, 2016). Special masters often lump GBS and CIDP together when the flu vaccine is at issue. *See*, *e.g.*, *Tomsky v. Sec'y of Health & Hum. Servs.*, No. 17-1132V, 2020 WL 5587365, at *5 (Fed. Cl. Spec. Mstr. Aug. 24, 2020) ("[F]or purposes of this decision I merely assume but do not decide that petitioner has established a medical theory causally linking the flu vaccine to CIDP"); *Strong v. Sec'y of Health & Hum. Servs.*, No. 15-1108V, 2018 WL 1125666, at *20 (Fed. Cl. Spec. Mstr. Jan. 12, 2018). I myself have done the same, motivated by a desire to adhere to past Program resolutions of such claims for the sake of judicial consistency. *Strong*, 2018 WL 1125666, at *20 (finding that the flu vaccine could cause CIDP).

But even if the leap from a flu vaccine-GBS connection to the flu vaccine being associated with CIDP is not as great as that for other vaccines, there are very few *reasoned* decisions linking

[16] Indeed, this causal evidence is the basis for a Table claim. 42 C.F.R. § 100.3.14 This means the Government has agreed that sufficiently-probative and reliable science on the topic exists to justify (in effect) conceding causation, at least for Program purposes. *Haskins v. Secretary of Health & Hum. Servs.*, No. 18-1776, 2020 WL 1870279 (Fed. Cl. Spec. Mstr. Mar. 13, 2020).

[17] By contrast, other kinds of Program claims involving conditions with multiple titles, all describing the same thing. Brachial neuritis, for example, is also referred to as Parsonage-Turner syndrome, or neuralgic amyotrophy—and therefore it makes no difference what term is used to characterize the diagnosis. *See Marshall v. Sec'y of Health & Hum. Servs.*, No. 21-1445V, 2024 WL 2059813, at *1 n.3 (Fed. Cl. Spec. Mstr. Apr. 12, 2024).

the flu vaccine to CIDP.[18] And I am aware of no such decisions addressing the claim that the flu vaccine worsened pre-existing CIDP, let alone the specific variant in question. *Jacunksi v. Sec'y of Health & Hum. Servs.*, No. 09-524V, 2014 WL 5168422, at *14 (Fed. Cl. Spec. Mstr. Sept. 23, 2014) (flu vaccine did not significantly aggravate CIDP; noting distinction between strength of evidence supporting flu vaccine-GBS association and flu vaccine-CIDP connection).

II. Petitioner Has Not Carried Her Burden of Proof

The first three *Loving* prongs in this case are met. The parties agree that Petitioner likely had anti-neurofascin CIDP prior to vaccination; she experienced progression of her disease course in the post-vaccination period; and her overall medical state could be said to have demonstrably worsened in comparison to her health pre-vaccination. But Petitioner's claim fails on the remaining three *Loving* prongs (which parallel the *Althen* prongs). I address them in the order of their significance.

Loving Prong Six (Althen Prong Three)

Dr. Nadareishvili forthrightly acknowledged that in this case (unlike the majority of claims alleging nerve damage attributed to a vaccine) he was *not* opining that the flu vaccine triggered the production of the anti-neurofascin antibodies responsible for Petitioner's illness. *See, e.g.*, Tr. at 18–19, 34–35, 69–70, 75. Indeed, since it is agreed Petitioner's injury *pre-dated* vaccination, he could not possibly show that the vaccine was responsible for what happened before its administration. He thus made no effort to show homology between amino acid sequences in the flu vaccine's antigenic components and protein components of neurofascin 155. *Id.* at 69, 70, 75.

Petitioner nevertheless contends that production of the specific *kind* of antibody in question associated with anti-neurofascin CIDP—an IgG4 antibody—could be *encouraged* due to vaccination (specifically via T-helper cells relevant to it). There are significant problems with this causation theory, but even if it were accepted as preponderantly established, the record in this case supports the conclusion that Petitioner's CIDP worsening would not likely occur as close-in-time to vaccination as appears to have happened in this case. The timeframe for worsening has not here been shown to be medically acceptable, measured from the date of vaccination.

Here, Petitioner's first post-vaccine neurologic symptoms occurred right around the date of vaccination (if not the same day, then a day or two later). Ex. 2 at 74; Ex. 3 at 105, 112. Regardless when symptoms worsening occurred, a process by which the flu vaccine—merely due to the immune system's initial innate response—would encourage a T-helper cell to in turn

[18] I have denied entitlement in cases where claimants attempted to link other vaccines with CIDP. *See, e.g.*, *Sanchez v. Sec'y of Health &Hum. Servs.,* No. 18-1012V, 2022 WL 1013264 (Fed. Cl. Spec. Mstr. Mar. 11, 2022) (Tdap vaccine not causal of CIDP).

stimulate B cells into producing more anti-neurofascin antibodies, or cause some other kind of illness stimulation, would take *several* days to occur at a minimum, even assuming immune memory relating to Petitioner's prior receipt of the flu vaccine was responsible for a faster reaction. *See also* Matiello Rep. at 14 (stating that "[i]t is immunologically implausible that the immunization caused an exacerbation of petitioner's CIDP symptoms given that the alleged exacerbation of petitioner's symptoms started on the same day of her flu immunization…"). And clinical symptoms would only come after that. Indeed, as Dr. Matiello credibly explained, the timeframe could be considerably longer than one week for all these interrelated processes to occur. Tr. at 116, 117, 121, 123–25. Thus, the timeframe in which Petitioner experienced additional post-vaccination CIDP symptoms pointed to as the start of worsening was far too short, post-vaccination, to reasonably implicate the flu vaccine as causal.

Loving Prong Four (Althen Prong One)

The record does not preponderantly support the conclusion that anti-neurofascin CIDP *could* be worsened due to receipt of a flu vaccine. Petitioner sought to establish that as a byproduct of receipt of the vaccine, whatever existing autoimmune process that was underway pre-vaccination due to the activity of anti-neurofascin 155 antibodies was heightened. But there are numerous deficiencies with the argument.

For example, Dr. Nadareishvili cited literature relating to the capacity of vaccines to cause autoimmune injuries via propagation of antibodies—and yet did not in this case attempt to show the flu vaccine could cause the production of anti-neurofascin antibodies (and even *disclaimed* that this had occurred, especially since the parties agree Petitioner's CIDP began before vaccination). Tr. at 18–19, 34–35. He contended vaccination generally could promote certain T-helper cells that might aid in the creation of these antibodies, via cytokine stimulation occurring as part of the innate immune process, but provided generalities about the effects of vaccination that were not linked to specific evidence bearing on the injury in this case. *Id.* at 32–33, 42–44, 83–84. He relied heavily on the mere fact that vaccination preceded Petitioner's worsening (despite case law firmly holding that a temporal association is not robust evidence of a *causal* association). *Id.* at 85, 90. And Dr. Nadareishvili's effort to link Petitioner's report of a shellfish allergic reaction pre-vaccination (as a model for the kind of IgG4 reaction associated with antibodies relevant to anti-neurofascin CIDP) was intellectually tantalizing, given the relevance of the IgG4 antibody both to allergic reactions and the likely disease process in this matter, but ultimately speculative.

In addition, as a general matter I did not find persuasive Petitioner's arguments that the impact of vaccination on the innate immune system (which first responds to the stimulation provided by a vaccine) can be pathogenic simply because it is known the innate response includes cytokine upregulation. *Olson v. Sec'y of Health & Hum. Servs.*, No. 13-439V, 2017 WL 3624085, at *20 (Fed. Cl. Spec. Mstr. July 14, 2017) (deeming it speculative to purport that cytokine

upregulation due to a vaccine "would be robust enough, and occur for long enough, to be pathogenic generally, let alone to cause" the complained-of injury), *mot. for review den'd*, 135 Fed. Cl. 670 (2017), *aff'd*, 758 F. App'x 919 (Fed. Cir. 2018). Without some other evidence also linking a disease process to innate immune aberrance, those kind of arguments over-rely on what is known about vaccine-immune system interaction to tell a story of disease pathogenesis that is ultimately speculative.

Dr. Matiello better demonstrated how the form of CIDP Petitioner experienced was unlikely to be influenced by the immune stimulation provided by a vaccine. As he noted, anti-neurofascin CIDP is not characterized as much by excessive inflammation (and corresponding immune-mediated damage, whether attributable to complement or other immune cells) harming the nerve myelin in the manner of other peripheral neuropathies like GBS, but instead could be viewed more accurately as a "nodopathy" that produces damage and resulting symptoms due to structural harm at the nerve nodes. Tr. at 108, 109. That kind of neuropathy may be antibody-mediated, but this does not mean that a vaccine is likely to encourage the creation or activity of such antibodies (and he noted the absence of support—in the literature or, to his knowledge, the neurologic community—for *any* vaccine as likely casual of this form of CIDP). *Id.* at 119–20, 128–29, 130, 136.

Dr. Nadareishvili's testimony did not successfully make up for the many components of his causation theory lacking independent reliable support. I am of course never compelled to embrace the *ipse dixit* of any expert's testimony, but may instead weigh its reliability and persuasiveness. *Legault v. Sec'y of Health & Hum. Servs.*, 177 Fed. Cl. 634, 650 (2025) (noting that "nothing requires a special master to accept an expert's conclusion "only by ipse dixit of the expert"). While Dr. Nadareishvili possessed the medical qualifications to opine on the issues in this case, he lacks specific expertise in the study of the relevant form of CIPD, or the different etiologies of peripheral neuropathies more broadly. And too much of his theory was based on plausibility or speculation, however well-reasoned it may have been. Dr. Matiello, in comparison, had expertise more congruent with the issues in dispute, and he convincingly and compellingly explained in what ways the proposed vaccine causation theory was inadequate. I reasonably gave more weight to Dr. Matiello's testimony.

Prong Five (Althen Prong Two)

Petitioner has not preponderantly established that her receipt of the flu vaccine likely *did* worsen her preexisting CIDP. To begin with, there is inadequate treater support for this conclusion. While *initially* treaters may have taken note of the temporal relationship between vaccination and her immediate clinical presentation, the records reveal they did so based on the supposition that they were looking at nascent GBS (which as noted above is reliably associated with the flu vaccine). *See, e.g.*, Ex. 3 at 47–48 (September 27, 2020 treatment—occurring a little more than

two weeks post-vaccination). Thereafter, as Petitioner's symptoms progressed, treaters favored CIDP as her diagnosis, but did not also continue to even speculate as to the flu vaccine's role in causing it.

Next, it is not evident from the totality of the record that the course of Petitioner's anti-neurofascin CIDP likely worsened *due to* vaccination (even if, for purposes of *Loving* prong three, I have concluded that Petitioner's health overall post-vaccination *was* worse).[19] Certainly Petitioner sought treatment for CIDP-like symptoms within three days of her vaccination, and she required IVIG and hospitalization. But the treatments she received proved effective (at least briefly), suggesting this was a transient incident. *See, e.g.*, Ex. 3 at 157, 170, 178, 194–203). It was not shown the impact of vaccination thereafter likely altered the course of her anti-neurofascin CIDP. Dr. Matiello also persuasively testified that comparable immune stimulation from *other* vaccines Petitioner received later did not similarly lead to relapse, undercutting the contention that the flu vaccine could have caused a reaction simply from its innate immune stimulation. Tr. at 143–45. (By contrast, Dr. Matiello maintained, treatment variations or difficulties in diagnosis leading to changes in medication *could* produce flares—and he credibly noted instances in the medical record where that appears to have occurred. *See id.* at 114). And he also convincingly opined (relying on his demonstrated familiarity with peripheral neuropathies like CIDP) that the overall course of the disease would involve relapsing/remitting symptoms, consistent with what Petitioner experienced. *Id.* at 126–27, 138–40.

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[19] Resolution of *Loving* prong five presents a conundrum, given its relationship with prong three. If it is determined that a person's pre-vaccination injury likely worsened post-vaccination under *Loving* prongs one to three, what does that mean for prong five? Of course, prong five requires linking the vaccine to the worsening—but what kind of evidence would do that job, or undermine the finding? In the *Althen* context, it is fair to look for objective evidence that a disease process was building up in the post-vaccination timeframe—proof of excessive inflammation leading to secondary symptoms, lab work corroborating overproduction of disease-causing antibodies, or treater speculation of a vaccine connection. Can that inquiry also be performed in the context of a significant aggravation claim, where the disease or condition is understood already to exist—and what would that evidence look like?

Complicating the analysis is the fact that the Federal Circuit has stated that it is improper to inquire into the *degree* of worsening, as if to do so is to require certainty as the evidentiary standard, rather than preponderance. *Osenbach*, 2025 WL 2387944, at *4. Yet at the same time, it would seem a significant aggravation claim *requires* consideration of whether a vaccine-instigated symptoms flare or relapse negatively alters the expected disease course, or simply causes a transient worsening that later subsides, without any greater impact on the disease's progress. Attempting to measure a post-vaccination worsening thus requires some frame of reference—a sense of what would have happened without vaccination—but it seems at present the Circuit disfavors performance of such a comparison.

One way to possibly resolve this dilemma would be to consider the issue from the perspective of the Act's six-month "severity" requirement. *See* Section 11(c)(1)(D)(i). All Program claims (Table and causation-in-fact, as well as significant aggravation claims) *must* demonstrate six months of post-onset sequelae. *Id.* In a case involving alleged vaccine aggravation of a preexisting illness or disease, the disease process is *always* likely to continue on more than six months after the alleged aggravating incident, making it appear as if severity is a given. But claimants must *still* show, where that incident is alleged to have been vaccine-related, that the six months or more of worsening *was* likely attributable to the vaccination. A transient effect (say, an aberrant immune response that caused a flare in symptoms but then ceased) would not meet the severity requirement, even if the vaccine unquestionably caused the symptom.

It is true that Petitioner's neuropathy symptoms did become more *evident* post-vaccination, and she arguably experienced a flare close-in-time to receipt of the flu vaccine that *could* reflect the impact of it on her immune system. But as Dr. Matiello established, that relapse was consistent with the expected course a person with CIDP of the kind Petitioner had would experience, rather than the by-product of vaccine-induced immune stimulation (even if the flu vaccine were deemed likely responsible for her most immediate CIDP symptoms). Thus, the vaccine cannot be said to be likely responsible for a worsening of Petitioner's disease course. Dr. Matiello also made some compelling points about the difficulty in identifying the diagnosis, and concomitant erroneous initial reliance by treaters on IVIg treatments (which later proved not all that ameliorative); these treatment issues could also have played a role in worsening her symptoms progression.

CONCLUSION

A Program entitlement award is only appropriate for claims supported by preponderant evidence. Here, Petitioner has not made such a showing. Petitioner is therefore not entitled to compensation.

In the absence of a motion for review filed pursuant to RCFC Appendix B, the Clerk of the Court **SHALL ENTER JUDGMENT** in accordance with the terms of this Decision.[20]

IT IS SO ORDERED.

/s/ Brian H. Corcoran
Brian H. Corcoran
Chief Special Master

[20] Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment if (jointly or separately) they file notices renouncing their right to seek review.